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Characteristics of Nonsmoking Women in NHANES I and NHANES I Epidemiologic Follow-up Study with Exposure to Spouses Who Smoke

Genevieve Matanoski, Sukon Kanchanaraksa, Deborah Lantry, and Yong Chang

Few studies have examined the relation between passive smoking and dietary intake in a large population. This report examines the nutrition and behavioral characteristics of 3,896 nonsmoking women from the first National Health and Nutrition Examination Survey (NHANES I) population in relation to exposure to environmental tobacco smoke. The data indicate that nonsmoking women who were exposed to husbands who smoked were more likely to be older, have lower education, live in the city, and have other health behaviors that could increase their risk of lung cancer compared with nonsmoking women with husbands who did not smoke. The nonexposed women were more likely to take vitamin supplements, to not drink alcohol, and to consume higher levels of dietary vitamin A, vitamin C, and calcium. The exposed and nonexposed women showed no difference in the levels of fatty acid intake nor in the levels of several other foods from the food frequency list after correction for age. Many of the differences that the authors observed between the women who were exposed and nonexposed to passive smoking could affect the risk of cancer. Therefore, they recommend that future studies of nonsmokers examine the influence of both passive smoking and diet on the risk of disease rather than examine the influence of a single factor. *Am J Epidemiol* 1995;142:149–57.

lifestyle; nutrition; smoking, passive; tobacco smoke pollution; women

Numerous studies have indicated that diet may influence the risk of lung cancer in smokers. In particular, it has been observed that high intake of beta-carotene or vitamin A is inversely related to the risk of this cancer (1–19). Other studies have examined the dietary characteristics of smokers and have found that their diet tends to be high in fats and low in fruits and vegetables (20–26). These data suggest that differences in diet might be related to the development of cancer. Not all studies have shown consistent results in regard to the effects of diet, especially when gender and level of smoking have been taken into consideration (2, 3, 6, 8, 16, 17). However, the observational data have been judged to be strong enough to warrant the initiation of two recent clinical trials of vitamin use in reduction of cancer incidence (27, 28); however, the results of these trials have been conflicting.

Exposure to environmental tobacco smoke has been reported to be associated with a risk of lung cancer in several studies and meta-analyses (29–37). The studies of passive smoking risks have often focused on women who report exposure to spouses who smoke. In fact, exposure in marriage is the primary source of passive smoke for women (38). While it would appear obvious that households might have similar dietary patterns in both spouses regardless of the individual smoking history of the husband or wife, few early papers reported examining the diet of wives who were exposed to passive smoking (35). Some investigators have tried to test the hypothesis that diet might play a role in the risk of lung cancer in nonsmokers but often without examining the possible exposure to passive smoking. The results of these studies have indicated that increases in beta-carotene intake and possibly other dietary factors in nonsmoking women may be associated with a decrease in the risk of lung cancer (39–41). These studies have a number of problems, including small numbers of subjects, lack of information on exposure to non-household sources of environmental tobacco smoke, and possible biases in the answers to dietary questions due to the presence of lung cancer in the cases. In addition, dietary data for cases often were collected from surrogates who had limited information about the eating habits of subjects (41). Among the studies that have examined the risk of lung cancer in association with passive smoking, two studies (42, 43) have controlled for dietary factors; when

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Abbreviations: CI, confidence interval; NHANES I, first National Health and Nutrition Examination Survey; OR, odds ratio.

From the Department of Epidemiology, Johns Hopkins University School of Hygiene and Public Health, Baltimore, MD.

Reprint requests to Dr. Genevieve Matanoski, Department of Epidemiology, Johns Hopkins University School of Hygiene and Public Health, 624 North Broadway, Room 280, Baltimore, MD 21205.

this was done, the authors reported that the risks associated with passive smoking remained. The recent multicenter study of the risk of lung cancer in nonsmoking women in relation to environmental tobacco smoke (44) showed no confounding from diet and thus the investigators did not control for this variable in reporting an association between passive smoking and lung cancer. However, two other recent studies (45, 46), which each included over 400 cases of nonsmokers, reported associations of lung cancer with diet but no influence of passive smoking on the risk estimates. Thus, the observed relations between diet, passive smoking, and lung cancer have not been consistent in all studies.

Because studies have indicated that dietary characteristics are associated with the smoking habits of subjects, some recent reports have investigated the relation between diets of nonsmokers and the smoking characteristics of household members (47, 48). These studies have suggested that nonsmoking individuals who were exposed to smokers may have different dietary patterns than persons who were not exposed to smokers. These studies usually were conducted in populations where there were small numbers of women exposed to tobacco smoke.

The current study of diets of nonsmoking women utilizes the large population that was included in the first National Health and Nutrition Examination Survey (NHANES I), which represented a sample of all noninstitutionalized individuals in the United States. This sample included persons from many socioeconomic, ethnic, and geographic groups with a variety of food habits and preferences from all parts of the United States. Individuals in this survey were generally healthy, and they answered questions about their diet with minimal bias related to disease. This should be an important population in which to investigate the question of what factors influence diet in nonsmoking women. It is a population that in the past has been widely used to examine nutritional questions.

MATERIALS AND METHODS

NHANES I, conducted between 1971 and 1975, surveyed noninstitutionalized individuals aged 1–74 years throughout the United States. The participants are representative of the US population, although there was a deliberate oversampling of children, elderly, women of childbearing age, and the poor in order to improve estimates of nutrition in groups who might be malnourished. A detailed 24-hour dietary questionnaire and a food frequency questionnaire that focused on the prior 3 months of food consumption was used in face-to-face interviews of the population. A total of 14,407 persons aged 25–74 years at the time of the first interview were included in the NHANES I Epidemiologic Follow-up Study in 1982–1984. The original survey asked about smoking information on only a small proportion of the participants. However, at the time of the follow-up interview 10 years later, information on smoking was obtained directly from the individual or from a surrogate in the event of a participant's death. At follow-up, all individuals were asked about marital status and whether they had ever been married to a spouse who smoked. The question did not specify the current vital status of that spouse or the amount he/she smoked. Thus, the question on spousal smoking encompasses the lifetime of the woman and no attempt was made to relate dietary data from NHANES I to active or passive smoking history. The study population for this report includes only the 6,890 females who reported that they had ever been married (table 1). From this group, those women who answered that they had never smoked 100 cigarettes or more in a lifetime were defined as nonsmokers (57 percent).

The data are derived from the public use tapes available from the National Center for Health Statistics. These tapes provide distributions of answers to specific questions on the food frequency questionnaire as well as summarized data on nutrients that are derived from the 24-hour diet recall survey. The National Center for Health Statistics derived the nutrient content of the diet using an adapted computer program that was developed at Tulane University and used in the Ten-State Nutrition Survey. The nutrient value of each food consumed as listed on the 24-hour recall diet was classified according to its values as reported in the US Department of Agriculture Handbook No. 8 (49) and from other sources. The datatape provides the summed values from the 24-hour food intake. The items include several vitamins, fats, carbohydrates, and some minerals. Retinoids and carotenoids were not reported. In addition, the food frequency questions were used in this study to identify selected food items that might represent healthy dietary behaviors.

NHANES I collected information on the use of supplemental vitamins in 1971–1974 using categories

TABLE 1. Ever married women by personal and husband's smoking history: first National Health and Nutrition Examination Survey (NHANES I), 1971–1975, and NHANES I Epidemiologic Follow-up Study, 1982–1984

Wife's smoking history	Husband's smoking history			Total
	Smoker	Nonsmoker	Unknown	
Smoker	2,477	480	21	2,978
Nonsmoker	2,424	1,393	79	3,896
Unknown	2	2	12	16
Total	4,903	1,875	112	6,890

of never, irregular, and regular (daily) use. Because many of the users took multivitamins, the current analysis did not try to account for variation in the intake of specific vitamins from these pills nor to include use of specific vitamins. The information on quantity and duration of vitamin taking was not part of the survey questionnaire. Thus, a cumulated dose could not be calculated.

The analyses examined the association between exposure to spousal smoking and diet and other characteristics in nonsmoking women using odds ratios calculated from maximum likelihood regression models. The odds ratios express the likelihood of the occurrence of the variable in women with spouses who smoked compared with that in women with spouses who did not smoke. The odds ratios were calculated first for the variable alone and then adjusted for age, and age and education, in separate models. Race was included in models with three confounding variables, but race did not add significantly to any model that included education. Variables were stratified into convenient approximately equal groups for analysis.

RESULTS

A total of 3,896 or 56.5 percent of the 6,890 married women indicated that they had never smoked 100 or more cigarettes in a lifetime, which classified them as nonsmokers. Exposure to spousal smoking was determined by the question, "Have you ever been married to someone who smoked cigarettes?" Smoking data were not verified in NHANES I or NHANES I Epidemiologic Follow-up Study with either biochemical markers or repeat questions for consistency. The data also do not provide any information on amount of smoking by the spouse and whether the exposure is current. Table 1 shows that 83.2 percent of smoking women and only 62.2 percent of nonsmoking women had spouses who smoked. In addition, the data indicate that 50.5 percent of wives of husbands who smoked and only 25.6 percent of wives of husbands who did not smoke are smokers. Thus, if there exists a misclassification of the nonsmoking status of the wives in this sample, it is likely that it occurred in the women with husbands who smoked, because these wives were twice as likely to be smokers than women who were married to men who did not smoke. In this study, as in other studies of exposure to environmental tobacco smoke, some of the differences in characteristics of nonsmokers attributed to the household exposure to smoke may actually be due to misclassification of women as nonsmokers in the smoking-exposed group.

Only the 3,896 nonsmoking women have been included in further analyses of factors associated with passive smoking. Table 2 indicates that age, race, socioeconomic factors, and residence are significantly associated with the likelihood of women being exposed to spouses who smoked. Women over 40 years of age are 1.3 to 1.4 times more likely to have been

TABLE 2. Demographic characteristics of nonsmoking wives by husband's smoking history: first National Health and Nutrition Examination Survey (NHANES I), 1971–1975, and NHANES I Epidemiologic Follow-up Study, 1982–1984

Characteristic	Husband's smoking history		Odds ratio*	95% confidence interval
	Smoker	Nonsmoker		
Age (years) at interview				
<40	707	489	1.00†	
40–59	777	391	1.39	1.17–1.64
≥60	940	513	1.29	1.10–1.51
Race				
Nonwhite	399	190	1.00†	
White	2,025	1,203	0.79	0.65–0.95
Education (years)				
<12	1,120	515	1.00†	
12	909	499	0.84	0.72–0.97
>12	382	366	0.48	0.40–0.57
Residence				
Rural	1,091	652	1.00†	
Urban	892	456	1.17	1.01–1.36
Other	435	285	0.91	0.76–1.09
Residence, adjusted for age and education				
Rural			1.00†	
Urban			1.24	1.07–1.44

* Odds ratio = $\frac{\text{odds of exposure to factor with smoking spouse}}{\text{odds of exposure to factor with nonsmoking spouse}}$.

† Referent group.

exposed to spousal smoking than are younger women. White women have a significantly lower probability of having a spouse who smoked than other racial groups in this population. This report uses education as a marker of socioeconomic status, because information on income was incomplete and because income would be a poor measure of social standing in women at older ages. An increasing level of education is associated with a decreased probability that women had been exposed to spousal smoking. For women with education beyond high school, the probability of exposure is only 48 percent of the likelihood for women who never graduated from high school. Women who lived in the city had a greater probability of exposure to passive smoke. This difference is not due to poverty in urban settings, because the odds ratio is actually increased from 1.17 (95 percent confidence interval (CI) 1.01–1.36) to 1.24 (95 percent CI 1.07–1.44) when both age and education are included in the model.

Low intake of fruit and vegetables and high intake of fats are considered to be related to an increased incidence of several cancers and heart disease. However, these diseases often are associated with poverty as well. To see whether any dietary factors are associated with smoking exposure, the data are presented with and without a correction for education as a measure of poverty. Because low education may be closely correlated with variables indicative of an unhealthy life-style, it might be considered inappropriate to correct dietary factors for education. That is, poverty itself is not the cause of disease, but poverty may lead to changes in life-style, such as poor diet, which is a more direct factor in the causative chain of disease. Adjusting the odds ratios for education would result in a dilution of the apparent effect of more direct causes of disease such as diet. Therefore, all data are presented using a model correcting only for age and then a model corrected for both age and education.

Several specific items were examined from the food frequency questionnaire on the presumption that the items would represent foods reportedly related to the risk of chronic diseases and those for which individuals could initiate preventive behavior such as lowering fat intake. The foods included one each from the vegetable and bread categories, one from dairy, and the others from meats. The items were total consumption of fried meats, lean ground meat, poultry with skin, whole grain bread, raw vegetables, and alcohol. Each food frequency item in the survey was not examined, because of the potential bias of utilizing multiple variables.

In the following tables showing the dietary items from the questionnaire, the data are from about 2,100 women with husbands who smoked and 1,200 women with husbands who did not smoke. Table 3 represents the distribution of dietary items for five of the six questions selected for analysis. Table 4 examines the association between eating low levels of raw vegetables and whole grain bread and eating high levels of fried foods and beef with high fat content and the likelihood of having exposure to passive smoking at home. While all of these less healthy eating practices of nonsmoking women are associated with living in a household with a husband who smoked, none of the odds ratios reaches significance when corrected for age and education except for consumption of beef. However, as shown in table 4, women who lived with spouses who smoked are significantly more likely to drink (odds ratio (OR) = 1.15, 95 percent CI 1.00–1.33). When they drank, the women who lived with a husband who smoked also drank more heavily than women who did not live with a husband who smoked. The risk of being exposed to passive smoking increases with increasing amounts of alcohol consumed. Women with husbands who smoked are more likely to answer that they eat the skin on the poultry (as a sign of increased fat intake) than do women whose husbands did not smoke (OR = 1.19, 95 percent CI 1.03–1.37). Nonsmoking women who took supplementary vitamins are less likely to have lived with a husband who smoked compared with women who did not take vitamins. All of these variables seem to show that nonsmoking women who never lived with a spouse who smoked have healthier dietary behaviors than women who have had a smoking spouse.

Table 5 examines the associations between nutrient levels from the 24-hour diet history and the presence

TABLE 3. Distribution of food frequency in subjects by spousal smoking status: first National Health and Nutrition Examination Survey (NHANES I), 1971–1975, and NHANES I Epidemiologic Follow-up Study, 1982–1984

Food item*	Husband's smoking status	
	Smoking	Nonsmoking
Raw vegetables		
Less than once daily	1,482	814
1 or more times daily	642	400
Fried foods		
Less than once weekly	1,162	686
1 or more times weekly	978	533
Type of beef		
Extra lean or none	563	364
Regular or lean	1,520	816
Whole grain breads		
Less than once daily	1,434	816
1 or more times daily	720	409
Eating skin on poultry		
No	1,068	651
Yes	1,051	553

* For each food item, different numbers of subjects provided answers.

TABLE 4. Health behavior of nonsmoking women in relation to spousal smoking: first National Health and Nutrition Examination Survey (NHANES I), 1971–1975, and NHANES I Epidemiologic Follow-up Study, 1982–1984

Characteristic/food	Adjusted for age only		Adjusted for age and education	
	Odds ratio*	95% confidence interval	Odds ratio*	95% confidence interval
Raw vegetables				
Less than once daily	1.00†		1.00†	
1 or more times daily	0.87	0.75–1.02	0.92	0.79–1.07
Fried foods				
Less than once weekly	1.00†		1.00†	
1 or more times weekly	1.10	0.95–1.26	1.05	0.91–1.21
Type of beef				
Extra lean or none	1.00†		1.00†	
Regular or lean	1.20	1.03–1.41	1.19	1.01–1.39
Whole grain breads				
Less than once daily	1.00†		1.00†	
1 or more times daily	0.96	0.83–1.12	1.01	0.86–1.17
Drinking				
No	1.00†		1.00†	
Yes	1.05	0.91–1.21	1.15	1.00–1.33
No. of drinks per day				
<2	1.00†		1.00†	
2–3	1.43	1.12–1.82	1.36	1.06–1.73
≥4	1.78	1.03–3.06	1.54	0.89–2.68
Eating poultry skin				
No	1.00†		1.00†	
Yes	1.19	1.03–1.37	1.14	0.99–1.33
Taking vitamin supplements				
Irregular or not	1.00†		1.00†	
Regular	0.82	0.71–0.95	0.86	0.74–1.00

* Odds ratio = $\frac{\text{odds of exposure to factor with smoking spouse}}{\text{odds of exposure to factor with nonsmoking spouse}}$.
An odds ratio greater than 1.0 means an increased proportion of spousal smoking.
† Referent group.

of a husband who smoked. A total of 2,007 women with husbands who smoked and 1,100 women with husbands who did not smoke answered these questions. For almost all items, women with greater nutrient intake are less likely to have had spouses who smoked. Only fatty acid intake shows no association between this nutrient and the probability of having a husband who smoked. For total calories and thiamine, although the odds ratios are below 1.00, increased dietary intake of either nutrient is not significantly associated with the probability of being exposed to a husband who did not smoke. The highest dietary intake of carbohydrates and riboflavin are associated with significantly low odds ratios of being exposed to a husband who smoked—0.77 (95 percent CI 0.61–0.97) and 0.70 (95 percent CI 0.52–0.94), respectively, after correction for age. The odds ratios are increased slightly and lose significance when corrected for both age and education. However, for three of the nutrients, calcium, vitamin A, and vitamin C, correction for age alone or correction for both age and education does not alter the significantly low probability that nonsmoking women with high intake of these nutrients are exposed to household tobacco smoke. At the highest category of intake, the odds ratios are significantly low. The fact that both vitamins A and C demonstrate an effect would suggest that intake of dietary fruits and vegetables is increased in women with husbands who did not smoke. The likelihood of a nonsmoking woman's exposure to a husband who smoked, as indicated by the odds ratio, decreases with increasing intake of calcium, carbohydrates, riboflavin, vitamin A, and vitamin C. However, after correction for both age and education, only vitamin C and possibly carbohydrates show a significant decrease in the probability of having a husband who smoked with an increase in exposure to the dietary variable.

The data in table 6 indicate that the weights of nonsmoking women are not influenced by the smoking status of their spouses, so that dietary differences are not simply due to variation in weight with passive smoking. However, their blood pressures show some suggestive differences. Both the systolic and diastolic

TABLE 5. Dietary nutrient intake of nonsmoking women from 24-hour recall data in relation to spousal smoking: first National Health and Nutrition Examination Survey (NHANES I), 1971–1975, and NHANES I Epidemiologic Follow-up Study, 1982–1984

Nutrient	Adjusted for age only		Adjusted for age and education	
	Odds ratio*	95% confidence interval	Odds ratio*	95% confidence interval
Calories (daily)				
≤1,000	1.00†		1.00†	
1,001–1,500	0.91	0.75–0.11	0.96	0.79–1.17
1,501–2,000	0.81	0.65–1.00	0.85	0.68–1.06
>2,000	0.79	0.61–1.01	0.85	0.65–1.10
p value (χ^2)	0.13		0.30	
p value (trend test)	0.16		0.40	
Carbohydrates (mgm/day)				
≤100	1.00†		1.00†	
101–150	1.10	0.87–1.39	1.13	0.90–1.43
151–200	0.86	0.68–1.08	0.91	0.72–1.15
>200	0.77	0.61–0.97	0.82	0.65–1.03
p value (χ^2)	0.004		0.02	
p value (trend test)	0.		0.06	
Fatty acids (mgm/day)				
≤10	1.00†		1.00†	
11–20	0.96	0.79–1.18	0.99	0.81–1.21
21–30	0.99	0.79–1.24	1.03	0.82–1.29
>30	0.95	0.74–1.22	0.99	0.77–1.28
p value (χ^2)	0.96		0.96	
p value (trend test)	1.00		0.998	
Calcium (mgm/day)				
≤300	1.00†		1.00†	
301–600	0.97	0.78–1.20	1.00	0.82–1.21
601–900	0.85	0.69–1.06	0.91	0.73–1.13
>900	0.74	0.59–0.92	0.80	0.63–1.00
p value (χ^2)	0.03		0.16	
p value (trend test)	0.05		0.23	
Thiamine (mgm/day)				
≤0.50	1.00†		1.00†	
0.51–1.00	0.97	0.80–1.17	1.02	0.84–1.24
1.01–1.50	0.85	0.67–1.08	0.93	0.73–1.18
>1.50	0.81	0.59–1.12	0.86	0.62–1.18
p value (χ^2)	0.26		0.54	
p value (trend test)	0.32		0.71	
Riboflavin (mgm/day)				
≤0.75	1.00†		1.00†	
0.76–1.50	0.81	0.68–0.98	0.85	0.70–1.02
1.51–2.25	0.77	0.62–0.97	0.82	0.65–1.03
>2.25	0.70	0.52–0.94	0.76	0.57–1.02
p value (χ^2)	0.04		0.18	
p value (trend test)	0.06		0.25	
Vitamin A (IU/day)				
≤1,500	1.00†		1.00	
1,501–3,000	0.91	0.74–1.12	0.95	0.77–1.18
3,001–4,500	0.91	0.72–1.27	0.98	0.80–1.30
>4,500	0.74	0.61–0.91	0.79	0.64–0.97
p value (χ^2)	0.02		0.10	
p value (trend test)	0.04		0.19	
Vitamin C (mgm/day)				
≤20	1.00†		1.00†	
21–50	0.84	0.67–1.06	0.88	0.69–1.11
51–80	0.82	0.64–1.05	0.87	0.68–1.12
>80	0.64	0.52–0.78	0.70	0.57–0.86
p value (χ^2)	0.0001		0.01	
p value (trend test)	0.0002		0.01	

* Odds ratio = $\frac{\text{odds of exposure to factor with smoking spouse}}{\text{odds of exposure to factor with nonsmoking spouse}}$.
An odds ratio greater than 1.0 means an increased proportion of spousal smoking.
† Referent group.

blood pressures of women are higher when they live in a home with environmental tobacco smoke. However, the odds ratios are not significant at any level for systolic blood pressure after correction for both age and education. Only a single odds ratio for diastolic blood pressure remains significant after correction for both age and education. However, because none of the data suggest a systematic increase in the probability of spousal smoking exposure with increasing blood pressure, there may be no relation between blood pressure and smoking exposure in the home. Blood pressure was selected to represent a condition that might not be

TABLE 6. Blood pressure and weight in nonsmoking women in relation to spousal smoking: first National Health and Nutrition Examination Survey (NHANES I), 1971–1975, and NHANES I Epidemiologic Follow-up Study, 1982–1984

Characteristic	Adjusted for age only		Adjusted for age and education	
	Odds ratio*	95% confidence interval	Odds ratio*	95% confidence interval
Systolic (mmHg)				
≤120	1.00†		1.00†	
121–140	1.25	1.04–1.49	1.19	0.99–1.42
141–160	1.30	1.04–1.63	1.22	0.97–1.53
>160	1.19	0.94–1.49	1.10	0.83–1.45
Diastolic (mmHg)				
≤70	1.00†		1.00†	
71–80	1.27	1.07–1.51	1.24	1.04–1.47
81–90	1.19	0.97–1.47	1.18	0.96–1.45
>90	1.46	1.28–1.66	1.37	0.98–1.92
Weight (lbs)‡				
<125	1.00†		1.00†	
125–149	0.99	0.80–1.22	0.96	0.78–1.19
150–174	1.07	0.86–1.33	1.00	0.80–1.25
>174	1.25	0.99–1.38	1.13	0.89–1.43

* Odds ratio = $\frac{\text{odds of exposure to factor with smoking spouse}}{\text{odds of exposure to factor with nonsmoking spouse}}$.
An odds ratio greater than 1.0 means an increased proportion of spousal smoking.
† Referent group.
‡ 1 lb = 0.45 kg.

treated adequately in women with poor preventive care. However, this marker may not provide a good indicator of a lack of medical care for a treatable condition, because, at certain blood pressure levels, all women, both those with and without health-seeking behaviors, may all receive treatment. This would mean that at higher levels of blood pressure almost all individuals might have blood pressure-lowering medicine and thus might have changed the apparent relation between the nonsmoking women's blood pressure and the presence or absence of spousal smoking.

DISCUSSION

The NHANES I and NHANES I Epidemiologic Follow-up Study databases provide an opportunity to examine the characteristics of nonsmoking women who lived in households with husbands who smoked and without husbands who smoked. The respondents' answers in these surveys should not be biased by the presence of disease as they might be in the traditional case-control study. Smoking questions asked in 1982–1984 might have been perceived as less threatening and produced less pressure on the women to provide socially acceptable answers than they would today. A problem with the study is that the dietary data were collected about 10 years before the smoking data and no attempt was made to relate the nutrient information to the smoking results in the surveys. Another serious problem with the study is that the smoking information from the respondents was not confirmed and therefore some of the "nonsmokers" may actually have been smokers. In this case, the misclassification may not be random. The error is more likely to occur for the wife married to a smoker than for a wife married to a nonsmoker. That is, the woman's nonsmoking status is more likely to be incorrect if she is married to a husband who smoked than if she is married to a husband who did not smoke. However, this type of error is common to most studies of passive smoking.

There is no information on other sources of tobacco smoke exposure in the home or at work, on the amount of smoking by the spouse, or on when in the woman's lifetime the exposure took place. We might note, however, that the question used to identify exposure in the home is similar to the questions used in many previous studies of passive smoking. In recent studies, investigators have attempted to validate smoking exposures with biochemical measures. They have also collected further data on the level of cigarette consumption in order to examine dose-response relations. Neither of these analyses was possible with these data.

Despite the lack of confirmation of the tobacco smoke exposure of the population, the NHANES I data have the major advantage of being from a very large study of nutrition in the total US population. The results of the analysis suggest that the characteristics of nonsmoking women who lived with spouses who smoked differed in many variables that could increase

their risk of chronic diseases, especially lung cancer. The nonsmoking women were likely to be less well educated, live in the city, engage less frequently in health-seeking behaviors, and consume lower levels of dietary nutrients that have been shown to have beneficial effects on disease risks, such as vitamin A and vitamin C. The demographic differences by exposure might have been expected. These characteristics are associated with smoking, and therefore the wife's demographic characteristics, by age, race, urban living, and poverty, would be expected to be similar to those of her husband who smoked. However, many of the effects, such as the differences in vitamin A and vitamin C intake, have remained significantly different in the women who were exposed to environmental tobacco smoke even after adjusting for age and education. Even for other variables, such as calcium intake, use of vitamin supplements, and abstinence from drinking, the differences remain at borderline significance after adjusting for both variables. Because diet may be a proximal factor in the causal chain of disease etiology that is related to low socioeconomic conditions, the persistence of these dietary effects after correction for education is noteworthy. In general, it is probably appropriate to correct only for age in order to avoid any overadjustment. Some of the relations between the variables may not be simple. Thus, exposure to household tobacco smoke may not represent just a single exposure but a complex of factors, many of which, such as low vitamin intake and high alcohol intake, have been shown to influence the risk of cancer.

Other studies have looked at the dietary intake of nonsmoking individuals in relation to the subjects' exposure to passive smoking, and similar dietary differences have been observed, particularly for beta-carotene and the history of exposure to tobacco smoke (47, 48). Sidney et al. (47) found a significantly lower beta-carotene intake in both men and women nonsmokers who were exposed to passive smoke ($p = 0.0001$). This difference was independent of demographic characteristics. The authors noted that a relative risk of 2 for the association of lung cancer and passive smoking would be reduced to 1.8 if adjusted for dietary carotene intake, and they stated that failure to take diet into account "may inflate estimates of the risk of passive smoking. . . by amounts that are modest, yet noteworthy" (47, pp. 1307–8). Le Marchand et al. (48) examined female nonsmokers and classified them by their exposure to passive smoking according to urinary cotinine levels. These investigators found an association with dietary vitamin A as well as with carotenoids and retinol. The intake was inversely related to the level of cotinine, and the trend was significant and unaffected by demographic characteristics. The data from NHANES I did not allow us to look specifically at carotenoids as a nutrient, but much of the vitamin A intake probably represents carotenoid intake.

Numerous studies have investigated the influence of diet on lung cancer in smokers (1–19). Diets that comprise low vegetable and low beta-carotene intake consistently have been shown to be associated with the risk of lung cancer. Sometimes a high fat intake also has been associated with a similar risk. However, few studies have attempted to evaluate diet in persons exposed to environmental smoke to determine whether diet plays a role in the development of cancer in these individuals as well. Because nonsmoking women are usually exposed to tobacco smoke in the home, the dietary and other characteristics of the wife should be similar to their husband who smokes. Several studies have included only women with lung cancer and have shown that there is a protective effect of beta-carotene and vegetables in the diet and beta-carotene in serum and lung cancer in women who did not smoke or who infrequently smoked (39–41). The risk of lung cancer from low intake of beta-carotene in recent studies in nonsmokers is as high as 2.5 to 5.0 (17, 39–41). This suggests that the risk from dietary factors is as high or higher as the reported relative risks for passive smoking from meta-analyses of combined studies, which have ranged from 1.3, 1.8, to 2.0 in recent papers (35, 36, 44). Yet, to our knowledge, few authors of studies of passive smoking have examined the influence of dietary factors (42–44) and few authors of studies of diet and lung cancer have addressed passive smoking in nonsmokers (45, 46). The results of five such studies (42–46) do not agree with those of two studies that have shown no effect of diet on the association between passive smoking and lung cancer (42, 44), two studies that have found no effect of passive smoking on the association between diet and lung cancer (45, 46), and one study that found independent effects of the two exposures (43). Further studies should investigate the role of both diet and passive smoking to determine the contribution of each in the development of lung cancer.

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REFERENCES

1. Bjelke E. Dietary vitamin A and human lung cancer. Int J Cancer 1975;31:397–405.
2. Gregor AG, Lee PN, Roe FJC, et al. Comparison of dietary histories in lung cancer cases and controls with special reference to vitamin A. Nutr Cancer 1980;2:93–7.
3. Shekelle RB, Lepper M, Liu S, et al. Dietary vitamin A and risk of cancer in Western Electric Study. Lancet 1981;2: 1185–9.
4. Kolonel NY, Nomura AM, Hinds MW, et al. Role of diet in cancer incidence in Hawaii. Cancer Res 1983;43(suppl): 2397s–2402s.
5. Stahelin HB, Rosel F, Buess E, et al. Cancer, vitamins, and plasma lipids: prospective Basel study. JNCI 1984;73: 1463–8.
6. Hinds MW, Kolonel LN, Hankin JH, et al. Dietary vitamin A, carotene, vitamin C and risk of lung cancer in Hawaii. Am J Epidemiol 1984;119:227–37.
7. Byers T, Vena J, Mettlin C, et al. Dietary vitamin A and lung cancer risk: an analysis by histologic subtypes. Am J Epidemiol 1984;120:769–75.
8. Samet JM, Skepper BJ, Humble CG, et al. Lung cancer risk and vitamin A consumption in New Mexico. Am Rev Respir Dis 1985;131:198–202.
9. Colditz GA, Branch LG, Lipnick RJ, et al. Increased green and yellow vegetable intake and lowered cancer deaths in an elderly population. Am J Clin Nutr 1985;41:32–6.
10. Wu AH, Henderson BE, Pike MC, et al. Smoking and other risk factors for lung cancer in women. JNCI 1985;74:747–51.
11. Menkes MS, Comstock GW, Vuilleumier JP, et al. Serum beta-carotene, vitamins A and E, selenium, and the risk of lung cancer. N Engl J Med 1986;315:1250–4.
12. Ziegler RG, Mason TJ, Stemhagen A, et al. Carotenoid intake, vegetables, and the risk of lung cancer among white men in New Jersey. Am J Epidemiol 1986;123:1080–93.
13. Middleton B, Byers T, Marshall J, et al. Dietary vitamin A and cancer—a multisite case-control study. Nutr Cancer 1986;8: 107–16.
14. Byers TE, Graham S, Haughey BP, et al. Diet and lung cancer risk: findings from the Western New York diet study. Am J Epidemiol 1987;125:351–63.
15. Goodman MT, Kolonel LN, Yoshizawa CH, et al. The effect of dietary cholesterol and fat on the risk of lung cancer in Hawaii. Am J Epidemiol 1988;128:1241–55.
16. Fontham ETH, Pickle LW, Haenszel W, et al. Dietary vitamins A and C and lung cancer risk in Louisiana. Cancer 1988;62:2267–73.
17. Le Marchand L, Yoshizawa CN, Kolonel LN, et al. Vegetable consumption and lung cancer risk: a population-based case-control study in Hawaii. JNCI 1989;81:1158–64.
18. Jain M, Burch JD, Howe GR, et al. Dietary factors and risk of lung cancer: results from a case-control study, Toronto, 1981–1985. Int J Cancer 1990;45:287–93.
19. Kvale G, Bjelke E, Gart JJ. Dietary habits and lung cancer risk. Int J Cancer 1983;31:397–405.
20. Witter FR, Blake DA, Baumgardner R, et al. Folate, carotene, and smoking. Am J Gynecol 1982;144:857.
21. Fehilg AM, Phillips KM, Yarnell JWG. Diet, smoking, social class, and body mass index in the Caerphilly Heart Disease Study. Am J Clin Nutr 1984;40:827–33.
22. Russell-Briefel R, Bates MW, Kuller LH. The relationship of plasma carotenoids to health and biochemical factors in middle-aged men. Am J Epidemiol 1985;122:741–9.
23. Stryker WS, Kaplan LA, Stein EA, et al. The relation of diet, cigarette smoking, and alcohol consumption to plasma beta-carotene and alpha-tocopherol levels. Am J Epidemiol 1988; 127:283–96.
24. Hebet JR, Kabat GC. Differences in dietary intake associated with smoking status. Eur J Clin Nutr 1990;44:185–93.
25. Enstrom JE, Kanim LE, Klein MA. Vitamin C intake and mortality among a sample of the US population. Epidemiology 1992;3:194–202.
26. Bolton-Smith C, Woodwork M, Brown CA, et al. Nutrient intake by duration of ex-smoking in the Scottish Heart Health Study. Br J Nutr 1993;69:315–32.
27. Blot WJ, Li J-Y, Taylor PR, et al. Nutrition intervention trials in Linxian, China: supplementation with specific vitamin mineral combinations, cancer incidence, and disease specific mortality in the general population. JNCI 1993;85:1483–92.
28. Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. N Engl J Med 1994;330:1029–35.
29. Trichopoulos D, Kalandidi A, Sparros L, et al. Lung cancer and passive smoking. Int J Cancer 1981;27:1–4.
30. Hirayama T. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. Br Med J 1981;282:183–5.
31. Correa P, Fontham E, Pickle LW, et al. Passive smoking and lung cancer. Lancet 1983;2:595–7.
32. Miller GH. Cancer, passive smoking and nonemployed and employed wives. West J Med 1984;140:632–5.
33. Garfinkel L, Auerbach O, Joubert L. Involuntary smoking and lung cancer: a case-control study. JNCI 1985;75:463–9.
34. Akiba S, Kato H, Blot WJ. Passive smoking and lung cancer among Japanese women. Cancer Res 1986;46:4804–7.
35. National Research Council. Environmental tobacco smoke: measuring exposures and assessing health effects. Washington, DC: National Academy Press, 1986.
36. Wald NJ, Nanchahal K, Thompson SG, et al. Does breathing other people's tobacco smoke cause lung cancer? Br Med J 1986;293:1217–22.
37. US Department of Health and Human Services. Reducing the health consequences of smoking: 25 years progress. A report of the Surgeon General. Rockville, MD: US DHHS, 1989. (DHHS publication no. (CDC) 89–8411).
38. Sandler DP, Helsing KJ, Comstock GW, et al. Factors associated with past household exposure to tobacco smoke. Am J Epidemiol 1989;129:380–7.
39. Pastorino V, Pisani P, Berrino F, et al. Vitamin A and female lung cancer: a case-control study on plasma and diet. Nutr Cancer 1987;10:171–9.
40. Koo LC. Dietary habits and lung cancer risk among Chinese females in Hong Kong who never smoked. Nutr Cancer 1988; 11:155–72.
41. Candelora EC, Stockwell HG, Armstrong AW, et al. Dietary intake and risk of lung cancer in women who never smoked. Nutr Cancer 1992;17:263–70.
42. Dalager NA, Pickle LW, Mason TJ, et al. The relation of passive smoking to lung cancer. Cancer Res 1986;46: 4808–11.
43. Kalandidi A, Katsouyanni K, Voropoulou N, et al. Passive smoking and diet in the etiology of lung cancer in nonsmokers. Cancer Causes Control 1990;1:15–21.
44. Fontham ET, Correa P, Reynolds P, et al. Environmental tobacco smoke and lung cancer in non-smoking women: a multicenter study. JAMA 1994;271:1752–9.
45. Alavanja MC, Brown CC, Swanson C, et al. Saturated fat intake and lung cancer risk among non-smoking women in Missouri. JNCI 1993;85:1906–16.
46. Mayne ST, Janerich DT, Greenwald P, et al. Dietary beta carotene and lung cancer risk in US nonsmokers. JNCI 1994; 86:33–8.
47. Sidney S, Caan BJ, Friedman GD. Dietary intake of carotene in nonsmokers with and without passive smoking at home. Am J Epidemiol 1989;129:1305–9.
48. Le Marchand L, Wilkens LR, Hankin JH, et al. Dietary patterns of female nonsmokers with and without environmental tobacco smoke. Cancer Causes Control 1991;2:11–16.
49. US Department of Agriculture. Composition of foods. Agriculture handbook no. 8. Washington, DC: US GPO, 1963.